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DIET IN DIABETES

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Despite the long recognition of diabetes as a clinical entity, no successful method of treating the disease existed until the nineteenth century, when Rollo and Bouchardat discovered independently the value of dietary restriction. The latter noted the disappearance of glycosuria in diabetics when food was scarce during the Siege of Paris, and applied that observation to the management of his patients. In the twentieth century, Frederick Allen's work (Allen, 1914, 1917, 1923; Allen and Sherrill, 1922) on undernutrition in experimental diabetes in dogs put the dietary treatment of diabetes on a more rational basis. The clinical results obtained by the use of restricted diets were good in the case of mild and obese diabetics, but among other diabetics deaths from diabetic coma and tuberculosis were very common. Pregnancy was an exceedingly rare occurrence, and diabetic children did not grow satisfactorily. The discovery of insulin by Banting and Best in 1921 revolutionized the treatment of the disease. Since its introduction the necessity for subcaloric diets, and indeed for dietary restriction of any kind, has become a matter of controversy.

Nobody questions the need for rigid dietary restriction for patients who are overweight, but for the diabetic who is not obese, and who requires insulin, three main types of dietary treatment are at present in use.

1. A strict regime, in which the calories and all the proximate principles are meticulously prescribed, and which aims at the maintenance of normal blood-sugar levels and of a practically sugar-free urine.

2. A more liberal method, involving the regulation of only the carbohydrate and total calorie intake, but allowing mild hyperglycaemia and glycosuria in order to prevent frequent insulin reactions.

3. A "free" diet, in which hyperglycaemia and glycosuria are disregarded, and in which the criteria of adequate insulin treatment are freedom from hunger, thirst, pruritus, nocturia, and ketosis, and the maintenance of weight and energy on the one hand, and the avoidance of hypoglycaemic symptoms on the other.

Such a diet, free of any restriction or regulation of food intake, was first advocated by Stolte (1931). It has been used by many physicians, including Lichtenstein (1938, 1945), Tolstoi and Weber (1939, 1940), Tolstoi, Almy, and Toscani (1942), Tolstoi (1943), and Micks (1943, 1944), and adopted independently by many

patients. The most important advantage claimed for this form of treatment is the psychological benefit obtained through freedom from irksome regulations. Diabetes is a chronic disease, at present incurable, and to minimize its hardships the fewer the restrictions imposed upon the patient's daily life the better. The advocates of "free" diets reason that if insulin is required at all it does not matter very much to the patient whether the dose is 30 or 50 units a day, whereas it does matter to him a great deal that he should be able to take a normal diet—"that" (as a diabetic doctor has said) "each meal should be an elegant satisfaction of appetite rather than a problem in arithmetic and a trial of self-abnegation" (*Practitioner*, 1946). They claim, too, that their patients are fully nourished and therefore more resistant to infections, including tuberculosis; that in children growth and sexual development are satisfactory; and that the incidence of pregnancy is high.

On the other hand, there are many who object that the use of liberal diets results in difficulty in clinical control; that frequent insulin reactions alternate with thirst, polyuria, and pruritus, associated with hyperglycaemia, glycosuria, and ketosis; that excessively large doses of insulin are required; that the patients become obese; and that degenerative complications occur earlier and more often.

This paper describes the study of a group of diabetics given a "free" diet over a period of five years.

Selection of Patients

Fifty patients were studied, of whom approximately one-half were newly discovered diabetics who had never been treated, and the other half patients who had been given dietary instructions at some time, but who were making no effort to adhere to them. Naturally the latter group included a number of rebellious spirits and a few people of subnormal intelligence. Obesity was the only bar to selection; as a result, the proportion of male patients included in the survey was unusually high. There were 34 males and 16 females. Their ages ranged from 9 to 71 years, and their occupations from sedentary work to heavy labouring.

A control group of 40 diabetics of comparable age and sex were chosen from out-patients attending the

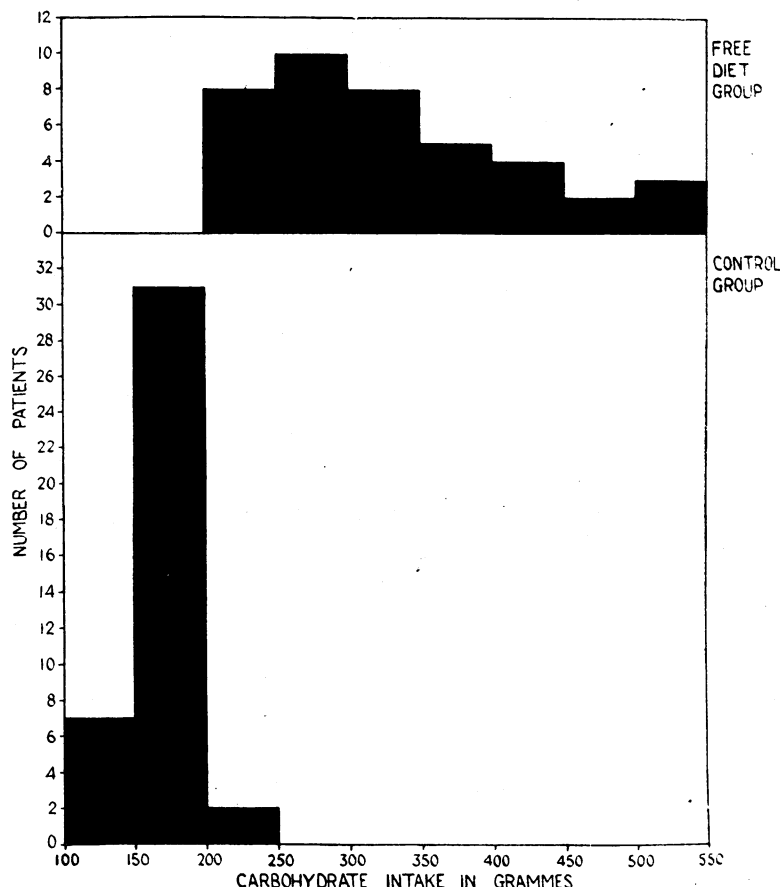


FIG. 1.—The daily carbohydrate intake of the "free diet" group and the control group.

Dietetic Clinic of the Royal Infirmary, Edinburgh. Only those who were adhering strictly to their diets and who required insulin were selected. Their diabetes was as well stabilized as could be expected of co-operative patients attending a busy centre. None of the patients in either group was suffering from any obvious diabetic complication or other disease at the time of selection.

In the charts that follow, the number of patients studied varies. This is not the result of deliberate selection, but of difficulties in the collection of data which are inseparable from clinical research on out-patients.

Method of Treatment

Patients who were taking a "free" diet were permitted to eat as their appetites dictated except for the omission of table sugar, jam, chocolates, and sweets, with the object of avoiding wild fluctuations of carbohydrate intake. During the period of study the British rationing system allowed all diabetics extra meat, bacon, milk, cheese, and fats. Thus the patients' standard of nutrition was probably higher than that of the general population.

Every patient received insulin once or twice a day. A simple powder method of testing the urine for acetone was used by the patients twice weekly on an

early morning sample of urine, and every day during intercurrent infections. It was the rule to report any positive tests to the clinic immediately. Testing for sugar was not encouraged, as glycosuria was to be expected.

Patients visited the clinic, on an average, once a month, but were seen more often during the course of infections or other emergencies. At every visit the weight was checked and any positive acetone tests, insulin reactions, nocturia, or other symptoms were recorded. The dose of insulin was adjusted from time to time to maintain an adequate weight and freedom from either diabetic or hypoglycaemic symptoms. On each occasion a quantitative analysis was made of the sugar content of two samples of urine: (1) a morning specimen passed twenty minutes after emptying the bladder on rising, before food or insulin had been taken; and (2) a 24-hour specimen. The collection was made on the previous day and the quantity measured in pints by the patient. A small sample of this collection was brought for examination. Though the results of these urine analyses for sugar were useful for our purposes, they were not used to regulate treatment. Changes in insulin dosage were dictated solely by the patient's weight and symptoms or by the development of ketonuria.

Diets

Most patients took three main meals in the day, with a snack in the morning and again in the evening before going to bed. Co-operative patients whose accuracy could be trusted were asked from time to time to keep a written record of everything they ate or drank over

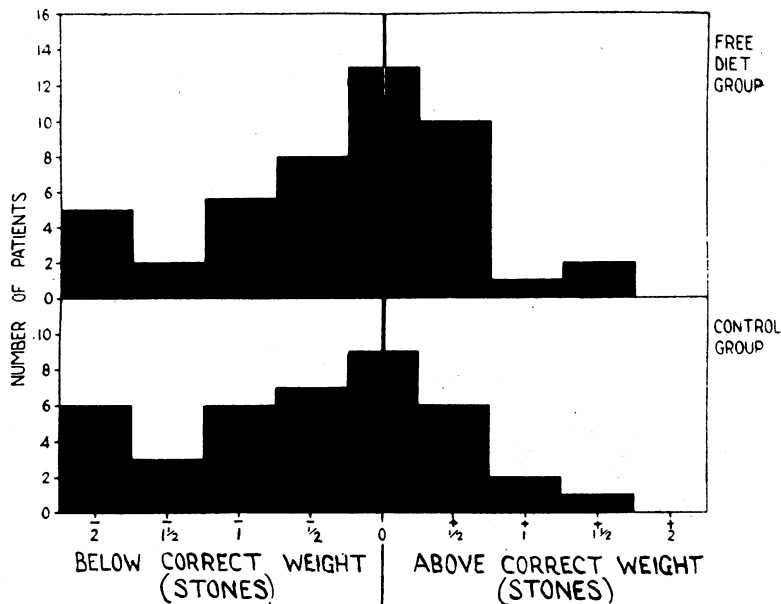


FIG. 2.—The relation of the present weights of the patients in both groups to their "correct" weights.

a period of one week. These records were discussed in detail with the patients, and it was felt that a reasonably accurate assessment of their diets was thus obtained. Analysis showed that the diets of the controlled group were higher in fat, much lower in carbohydrate, and somewhat lower in total calorie value than those of the group taking a "free" diet. Fig. 1 shows that the carbohydrate intake was considerably higher on a "free" diet than on the diets taken by the control group.

The carbohydrate intake on a "free" diet remained remarkably constant from one week-day to another, but rose or fell significantly at the week-end. The heavy labourer enjoying a lazy week-end was not so hungry, whereas the sedentary worker who played games ate more.

The carbohydrate intake of patients given a "free" diet from the time that their diabetes was diagnosed did not differ significantly from that of patients who had lapsed from a strict dietary regime. This eliminated the possibility that members of the latter group were unconsciously following dietetic instructions given in the past.

Weights

If the criticism often levelled at the use of "free" diets, that many of the patients become obese, were true, it would constitute a grave objection to this form of treatment.

Fig. 2 compares the present weights of the patients after some five years of treatment with their "correct" weights. The distribution is remarkably similar for the two groups, there being no increased tendency for those taking a "free" diet to become obese. The patients who did become overweight on a "free" diet

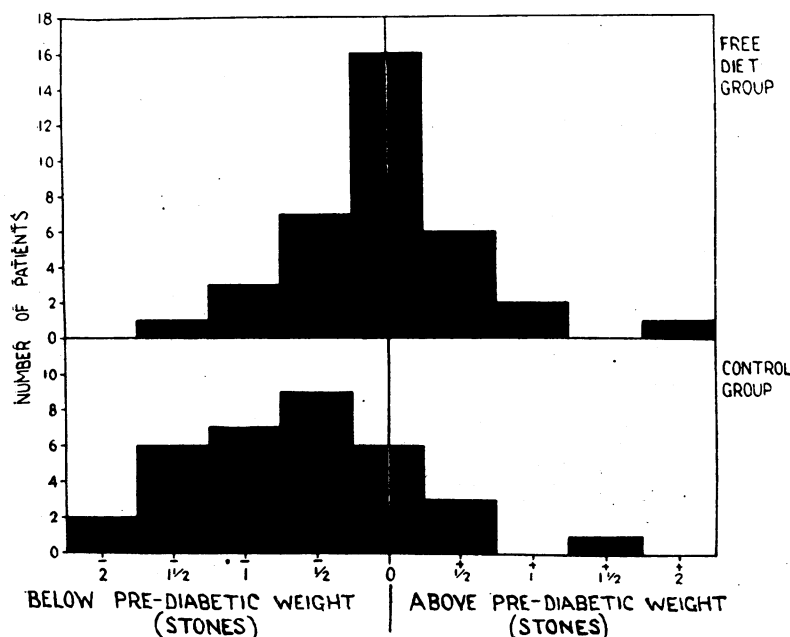


FIG. 3.—The relation of the present weights of the patients in both groups to their pre-diabetic weights.

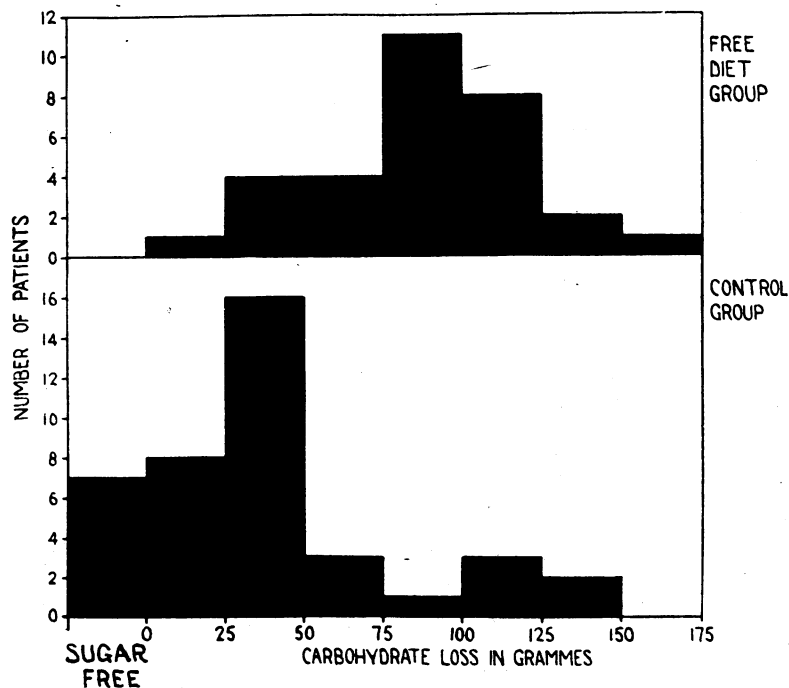


FIG. 4.—The daily carbohydrate loss in the urine of the patients in the "free diet" and control groups.

all gave a history of obesity in the past, though as a result of uncontrolled diabetes they were of normal or subnormal weight when they appeared for treatment.

Fig. 3 shows the relation of the patients' present weights to their "average" weights—that is, the weights which they maintained before the onset of diabetes. It is clear that the tendency of patients taking a "free" diet to revert to their pre-diabetic weight held not only for those who were previously obese but for the group as a whole. Dietetically controlled patients, on the other hand, tended to remain below their pre-diabetic weight.

If, as is generally agreed, a normal weight is the therapeutic ideal, patients who give a history of obesity before the onset of diabetes, even if they are thin by the time they appear for treatment, should be given controlled diets. Those who have never been overweight may be given full diets without much danger of their becoming obese.

Urine-sugar Studies

The accurate collection, once a month for six consecutive months, of the urine passed during the 24-hour period of a working day demands the full co-operation of the patient. Only those who could be relied upon to be accurate are included. The results shown in Figs. 4 and 5 are based on an average of the sugar content of six 24-hour samples. Meal charts covering the 24-hour periods during which the urine collections were made were brought with each specimen.

The average daily carbohydrate loss in the urine was greater in the "free diet" group than in the control group

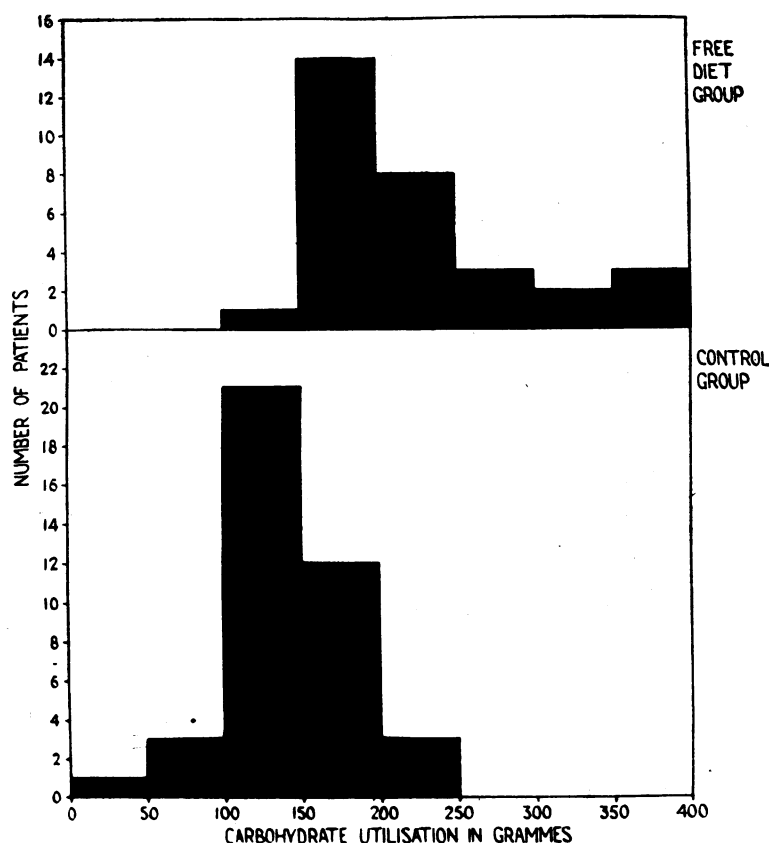


FIG. 5.—The daily carbohydrate utilization of the patients in the "free diet" and control groups.

by 50 to 75 g. (Fig. 4). In spite of this (Fig. 5) their total daily carbohydrate utilization was greater by at least 50 g. because of their higher carbohydrate intake. It is evident, therefore, that the quantity of carbohydrate metabolized by these patients on a "free" diet was adequate.

Blood-sugar Studies

Information about blood-sugar levels was obtained by taking blood samples from co-operative members of both groups in their homes or places of work during a normal day. The samples were taken before breakfast, lunch, and the evening meal. A fasting period of at least two hours preceded the withdrawal of blood. The highest of these three blood-sugar recordings was used in constructing Fig. 6, which shows that a higher proportion of patients on a "free" diet exhibited hyperglycaemia and that the degree of hyperglycaemia was greater than in the case of the control group. However, more than half of the patients in the latter group had blood sugars above 200 mg. per 100 ml. before a meal, illustrating how difficult it is to attain the ideal of normoglycaemia even when postprandial rises in blood sugar are discounted.

The lowest blood sugars were similar in the two groups. From these findings it follows that the fluctuation of blood-sugar level during the course of the day was greater in the group of patients taking a "free" diet.

Clinical Control

Eleven of the original 50 patients given a "free" diet could not be satisfactorily controlled with this form of treatment. They are considered in detail below. By adjusting their insulin dosage from time to time no difficulty was experienced in keeping the remaining 39 patients free from hunger, thirst, pruritus, and nocturia. Ketonuria was only an occasional finding during the course of intercurrent infections. The frequency of minor insulin reactions was not appreciably higher than among dietetically controlled patients. Like all diabetics, most of the patients experienced mild reactions from time to time, which were readily alleviated by taking a lump of sugar. (Hypoglycaemic coma is considered later.) The patients maintained an adequate weight and full physical energy. They seemed happy, and had a balanced outlook on their disease. Intercurrent infections did not appear to be commoner than among the control group.

The incidence of pregnancy was high. Seven of the eight married women in the child-bearing age became pregnant, one woman twice during the period studied. Of the eight babies, six are alive and two were stillborn. It has been noted that a high proportion of the women reporting to the Diabetic Pregnancy Clinic were taking a diet of normal calorie value at the time of conception, either because they belonged to the "free diet" group or because they had defaulted from their dietetic instructions.

There were seven young people in the "free diet" group of patients. Three were girls of 16 in whom menstruation had begun before the onset of diabetes, and who had stopped growing. The other four were boys of 15 or under in whom further growth was to be expected. Details of their rate of growth are given in Table I. It is significant that growth, which had been unusually rapid before the onset of diabetes, continued

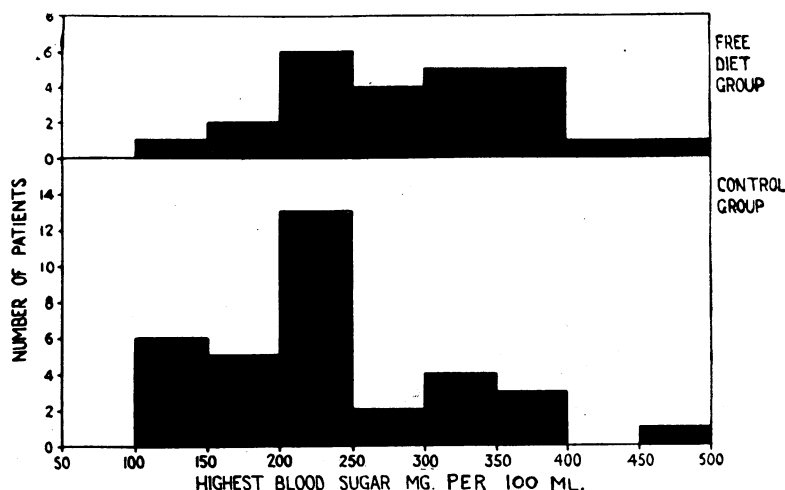


FIG. 6.—The highest blood-sugar levels recorded in patients of both groups.

at an excessive rate while they were taking a "free" diet.

TABLE I.

| Case | Age at End of "Free Diet" Treatment | Duration of "Free Diet" Treatment | Increase in Height During Treatment | Normal Increase in Height for Same Period |
|------|-------------------------------------|-----------------------------------|-------------------------------------|---|
| 1 | 11 years | 2 years | 4.5 in. (11.4 cm.) | 3.8 in. (9.7 cm.) |
| 2 | 17 " | 3 " | 7.5 in. (19.1 cm.) | 4.6 in. (11.7 cm.) |
| 3 | 16 " | 2 " | 6.5 in. (16.5 cm.) | 3.2 in. (8.1 cm.) |
| 4 | 15½ " | ½ year | 1.5 in. (3.8 cm.) | 0.8 in. (2.0 cm.) |

Normal height increments are taken from the table on page 290 of *Endocrine Disorders in Children and Adolescents*, by Le Marquand and Tozer, London, 1943.

Coma

Among the 50 patients on a "free" diet observed over five years three incidents of diabetic coma and seven of hypoglycaemic coma occurred. This contrasts with five incidents of diabetic coma and four of hypoglycaemic coma among the 40 patients in the control group. The incidence of hypoglycaemic coma is a little higher in the "free diet" group, and perhaps reflects the greater risk which attends an unregulated food intake and large doses of insulin.

Insulin

The type of insulin required and the number and timing of the injections varied from patient to patient. The majority were stabilized on an injection of soluble insulin and one of zinc-protamine insulin half an hour before breakfast, and a second dose of soluble insulin half an hour before the evening meal: 40 of the 50 patients required two injections a day.

TABLE II.

| Total No. of Patients | No. Requiring More Insulin | No. Requiring Less Insulin | No. Requiring the Same Dose |
|-----------------------|----------------------------|----------------------------|-----------------------------|
| 47 | 20 | 4 | 23 |

Table II shows the changes in insulin requirement of the patients on a "free" diet. The dose required after five years' treatment or in the case of the 11 failures at the time of changing to a controlled diet is compared with the dose needed three months after beginning this form of treatment. The insulin requirement was considered to be unchanged if the rise or fall did not exceed 20% of the original dose. Only 47 patients are included in the table, as three defaulted from the clinic. The insulin requirement of the group as a whole has tended to rise during treatment. The most striking increases occurred in children of puberty age and young adults. Whether this trend will continue, making further changes to controlled diets necessary, is as yet uncertain.

Repeated blood-sugar curves were obtained in one-third of the patients in an attempt to assess whether the diabetic state was becoming milder or more severe under "free diet" treatment. In general, the results obtained agreed with the trend shown by the comparative insulin requirements—namely, an apparent increase in severity.

Complications and Failures

Miscellaneous illnesses, including pneumonia, cystitis, and infective hepatitis, occurred in a few patients, but only complications known to be associated with diabetes are considered in detail.

No patient in the group has so far developed peripheral arteriosclerosis, hypertension, or albuminuria. Two patients have suffered temporarily from diabetic neuropathy. Diabetic retinopathy has developed in four patients, and senile cataract in one. It is interesting to note that one young man developed diabetic retinopathy at the age of 28, only one year after becoming diabetic. It was definitely known that he had not been suffering from diabetes previously. In contrast, two patients who had never paid any attention to dieting since the onset of the disease showed no sign of degenerative complications after a duration of diabetes of 23 years in the case of a woman who is now 27, and of 25 years in the case of a man now aged 45.

During the course of this five-year survey 11 patients have had to be changed from "free" diets to regulated diets. In seven of them the diabetic state became too severe to be satisfactorily controlled with a total daily dose of 80 units of insulin. It was considered unjustifiable to raise this dosage further. Five of the seven were children and two were young adults. With regard to the other four, frequent insulin reactions made it essential to regulate the diets of one adult and one child; and two women were given subcalorie diets because they became obese and began to suffer from pruritus vulvae. Both had been overweight before the onset of diabetes. After the change to a restricted diet the two obese women improved immediately. Of the two patients in whom frequent insulin reactions dictated the change, one improved; the other, a child, remained liable to frequent hypoglycaemic attacks. There was a considerable fall in the insulin requirement of the seven patients who had been uncontrollable while taking 80 units a day on a "free" diet. A close study of the growth curves of the growing children revealed that the very rapid growth which took place while they were on a "free" diet was not maintained after the change to a more restricted regime, though the rate was still satisfactory.

Discussion

From our experience of a group of 50 diabetics given liberal diets and insulin over a period of five years we are satisfied that, if adolescents and obese diabetics are excluded, clinical control, as defined earlier in this paper, can be attained in most patients. However, the degree of hyperglycaemia and glycosuria and the daily fluctuation of blood-sugar levels are undoubtedly greater in such patients than in those on controlled diets.

Those diabetics whose insulin requirements gradually rose while taking the "free" diet until 80 units were required daily showed an increased liability to ketosis when infections supervened. Moreover, the danger of insulin reactions was increased roughly in proportion to the dose of insulin employed. We feel, therefore, that it is unjustifiable to keep patients on a "free" diet when such large doses are needed for clinical control. Seven patients have had to be changed to controlled diets for this reason, and the change will almost certainly be necessary for two other patients in the near future. Of these nine, five are adolescent children and the remaining four are young adults between the ages of 20 and 35.

The need for dietary restriction has become apparent after periods on liberal diets ranging from six months to five years. Fortunately, when a change over to a controlled diet has been made the insulin requirement has shown a considerable fall, and no permanent ill

effect has been observed. It is uncertain whether increasing insulin requirements may make dietary restriction necessary for other patients in years to come, but the fact that failures are still appearing after five years' treatment is disquieting. Results with non-obese patients aged over 35 have been uniformly good, and it may well be that it is for this group of diabetics that "free" diets will have their greatest value.

There are many physicians who believe that continued hyperglycaemia increases the severity of the diabetic state, and that if it is prolonged the development of the degenerative complications of diabetes is facilitated. However, a normal blood-sugar concentration may exist when subcalorie diets are used in the presence of inadequate quantities of liver and muscle glycogen, while hyperglycaemia by no means necessarily implies inadequate stores unless there is an associated ketosis. Indeed, Peters and Van Slyke (1931) defined diabetes mellitus as an inability to store glycogen, and made no mention of hyperglycaemia. It is important to distinguish hyperglycaemia which is endogenous in origin, due to breakdown of glycogen stores and tissue protein—an expression of poor carbohydrate utilization and usually accompanied by ketosis—from hyperglycaemia of exogenous origin resulting from an abundant carbohydrate intake, with which, however, carbohydrate utilization as the result of insulin therapy is adequate and there is no associated ketosis. This distinction is rarely made, and the evil effects of uncontrolled diabetes are often attributed to the hyperglycaemia rather than to the poor utilization of carbohydrate. No one questions the undesirability of hyperglycaemia of endogenous origin, but considerable controversy exists on the harmfulness of exogenous hyperglycaemia.

It thus becomes important to inquire into the relationship of hyperglycaemia to the pathological physiology of diabetes. The production of diabetes by pancreatectomy in the laboratory of von Mering and Minkowski (1889), and the discovery of insulin with its dramatic therapeutic properties, placed the pancreas in the forefront of diabetic pathology. In 1922 Allen and Sherrill demonstrated that the pathological changes in the β -cells of the islets of Langerhans caused by partial pancreatectomy in dogs could be accentuated by giving the animals high-calorie diets. Further, Dohan and Lukens (1947) reported that normal cats could be made diabetic when a sustained hyperglycaemia was induced by repeated intraperitoneal injections of glucose. There is thus ample evidence that in experimental animals the insulin-producing cells of the pancreas can be damaged by hyperglycaemia.

In man, however, diabetes cannot be considered purely as a disease of the pancreas. In the first place, a histologically normal pancreas is found at necropsy in a considerable proportion of diabetics (Warren, 1930), and, furthermore, the complete removal of the human pancreas causes a relatively mild form of diabetes, readily controlled by not more than 40 units of insulin a day (Ricketts, 1947), rather than the grave metabolic disorder often seen in the spontaneously occurring disease, for the control of which much larger doses of insulin may be required. It seems certain that in human diabetes, at least in its severer forms, other endocrine glands are involved. Animal experiments have shown the importance of the anterior pituitary gland and of the suprarenal cortex in the pathology of the disorder.

Houssay (1936) discovered that depancreatized dogs lived longer if the anterior pituitary was also removed. The reverse of this—namely, that permanent diabetes could result from continued injections of anterior pituitary extracts in adult dogs—was demonstrated by Young (1937). Along similar lines, Long and Lukens (1934) found that adrenalectomy ameliorated pancreatic diabetes in the cat, and Long and his co-workers (1940) and Ingle (1940) demonstrated the aggravation of the diabetes of partially depancreatized rats by the administration of certain fractions of suprarenal cortical extract. Further support is provided by clinical evidence: the occurrence of diabetes in association with acromegaly and Cushing's syndrome; and the striking fall in insulin requirement which occurs in the rare event of the development of Addison's disease in a diabetic.

However, in most cases of diabetes in man it is uncertain to what extent the syndrome is due to failure of insulin production by the pancreas, and to what extent to an abnormal inhibition of the action of insulin by the anterior pituitary and cortical hormones. The effect of low-calorie diets in lessening the severity of human diabetes is an undoubted clinical fact, demonstrated on a large scale in the countries of Western Europe during the German occupation, but we do not know whether it is the result of "sparing" the pancreas or of inhibiting anterior pituitary function.

Jacobs (1948) showed that prisoners of war who had been suffering from prolonged undernutrition presented many of the features of anterior pituitary depression; and it is well known that the clinical picture of advanced anorexia nervosa is in many respects indistinguishable from Simmonds's disease. Any attempt to alleviate diabetes by depressing the secretion of the "diabetogenic" hormone of the anterior pituitary, however, must lead to simultaneous suppression of the growth, thyrotrophic, corticotrophic, and gonadotrophic hormones. Indeed, the recent work of Young *et al.* (1949) has strongly suggested that, at least in animals, the growth and "diabetogenic" hormones are identical. From clinical observations Lawrence (1949) has formed the concept of "total diabetes." He believes that such a state is reached by most young diabetics within three years of the onset of their disease. Their daily insulin requirement becomes stable at between 40 and 60 units. His interpretation of this state is a final cessation of insulin secretion by the pancreas. If this view is correct the steadily rising insulin requirement of some young diabetics cannot be attributed to further depression of insulin secretion, but must be due rather to inhibition of injected insulin by pituitary or suprarenal cortical hormones.

The rapid growth, advanced bone age, and precocious dental development of children at the onset of diabetes (White, 1946) are generally regarded as expressions of overactivity of the anterior pituitary gland. It is significant that in our group of patients the children at the age of puberty maintained an accelerated growth while taking a "free" diet, and also showed the most striking increase in insulin requirement. Further, these tendencies were not maintained after a change to a controlled diet had been made. It is possible that the underlying pituitary overactivity of these children was perpetuated by the metabolic stimulus of a high calorie intake. The same mechanism may be responsible for the apparent worsening of the diabetic state of some of the young adult patients, and for the high incidence of pregnancy.

If this view is accepted, it follows that, for the young diabetic at least, a diet which is generous enough to allow for adequate growth and sexual function will to some extent result in stimulation of the anterior pituitary gland and a consequent worsening of the diabetic state.

Opinion is conflicting about the relationship of hyperglycaemia to peripheral vascular disease. Joslin (1947), as a result of his vast experience, is convinced that the meticulously controlled patient is "the one in best condition" after 20 years of diabetes. White and Waskow (1948), in a review of 350 young adults who had been diabetic for at least 20 years, showed that there was a correlation between the severity of vascular disease on the one hand and *poor diabetic control*, as shown by hepatomegaly, incidence of diabetic coma, hypercholesterolaemia, and hyperglycaemia, on the other. The longest follow-up of a group of patients taking a "free" diet, by Lichtenstein, indicated that the incidence of degenerative complications after 10 years' treatment was not unduly high, but even this period of observation is too short for a reliable assessment to be made. Dolger (1947), who observed 200 diabetics over a period of 25 years, found that vascular disease occurred neither sooner nor more frequently in patients with persistent glycosuria than in those whose urine was kept sugar-free. He believed that complications occurred more often in the older patients, and that the duration of the disease was the most important factor. A similar conclusion was reached by Goodof (1945) in relation to the characteristic renal lesion of diabetes first described by Kimmelstiel and Wilson (1936). As for diabetic retinopathy, Ballantyne (1946) stated: "The outstanding paradox is the lack of any apparent relation between the severity of the diabetes, the control of the condition by treatment, and the presence or absence of retinal changes." Ballantyne's opinion is endorsed by Wagener (1945) and by Croom and Scott (1949).

Rundles (1945) gave a detailed report of his observations in 125 cases of diabetic neuropathy. He found that in most instances neuropathy followed a long period of grossly neglected diabetic management associated with hyperglycaemia, thirst, polyuria, and loss of weight.

While the correlation between the development of some of the degenerative complications of diabetes and poor control of the disease is at least suggestive, no one has yet produced evidence that hyperglycaemia, accompanied by adequate carbohydrate utilization and good clinical control, is responsible for the development of vascular complications. The present survey can make no contribution to the solution of this problem, as five years is too short a period on which to base conclusions.

Summary

An investigation of the use of a "free" diet in the treatment of 50 diabetics over a period of five years is described. For comparison, 40 dietetically controlled patients were observed over the same period.

Clinical control, as defined in the paper, was achieved in 39 of the 50 patients. Though hyperglycaemia and glycosuria were the rule, carbohydrate utilization was satisfactory. The incidence of pregnancy was high, and the growth of the children was unusually rapid.

Results were uniformly good in patients over the age of 35, but the insulin requirement of the younger patients tended to rise. Five children and two young adults showed

a progressive worsening of their diabetic state, and had to be given controlled diets for this reason. The change was also necessary for two patients who suffered from frequent insulin reactions, and for two others who became obese and developed pruritus. Only those patients who gave a history of obesity before the onset of diabetes became overweight while taking a "free" diet. There were three episodes of diabetic coma and seven of hypoglycaemic coma among the 50 patients during the five-year period of study; these did not vary very significantly from similar incidents among the controlled group.

The relationship of hyperglycaemia to the pathological physiology of diabetes and to the development of degenerative vascular complications is discussed. A distinction is made between "exogenous" hyperglycaemia, which accompanies a high carbohydrate intake in patients whose carbohydrate utilization is adequate, and "endogenous" hyperglycaemia, which results from breakdown of glycogen stores and tissue protein in association with poor control of the disease.

While there is no evidence that "exogenous" hyperglycaemia is the cause of degenerative complications, there is reason to believe that the high-calorie diets with which it is associated may be responsible for increasing the severity of the diabetic state in children and young adults. A possible mechanism of this action is suggested.

REFERENCES

- Allen, F. M. (1914). *J. Amer. med. Ass.*, **63**, 939.
 — (1917). *Amer. J. med. Sci.*, **153**, 313.
 — (1923). *J. metab. Res.*, **3**, 61.
 — and Sherrill, J. W. (1922). *Ibid.*, **2**, 803.
 Ballantyne, A. J. (1946). *Trans. Ophthal. Soc. U.K.*, **66**, 503.
 Banting, F. G., and Best, C. H. (1922). *J. Lab. clin. Med.*, **7**, 464.
 Croom, J. H., and Scott, G. I. (1949). *Lancet*, **1**, 555.
 Dohan, F. C., and Lukens, F. D. W. (1947). *Science*, **105**, 183.
 Dolger, H. (1947). *J. Amer. med. Ass.*, **134**, 1289.
 Goodof, I. I. (1945). *Ann. intern. Med.*, **22**, 373.
 Houssay, B. A. (1936). *New Engl. J. Med.*, **214**, 971.
 Ingle, D. J. (1940). *Proc. Soc. exp. Biol., N.Y.*, **44**, 176.
 Jacobs, E. C. (1948). *J. clin. Endocrinol.*, **8**, 227.
 Joslin, E. P. (1947). *Med. Clin. N. Amer.*, **31**, 259.
 Kimmelstiel, P., and Wilson, C. (1936). *Amer. J. Path.*, **12**, 83.
 Lawrence, R. D. (1949). *Lancet*, **2**, 401.
 Lichtenstein, A. (1938). *J. Pediatr.*, **12**, 183.
 — (1945). *Acta pediat.*, **32**, 556.
 Long, C. N. H., Katzin, B., and Fry, E. G. (1940). *Endocrinology*, **26**, 309.
 — and Lukens, F. D. W. (1934). *Science*, **79**, 569.
 von Mering and Minkowski (1889). Quoted from *The Treatment of Diabetes Mellitus*, edited by E. P. Joslin and others, p. 86, 8th ed., 1946. London.
 Micks, R. H. (1943). *British Medical Journal*, **1**, 598.
 — (1944). *Ibid.*, **2**, 784.
 Peters, J. P., and Van Slyke, D. D. (1931). Quoted by Tolstoi (1943).
 Ricketts, H. T. (1947). *Med. Clin. N. Amer.*, **31**, 267.
 Rundles, R. W. (1945). *Medicine*, **24**, 111.
 Stoltz, K. (1931). *Med. Klin.*, **27**, 831.
 Tolstoi, E. (1943). *Amer. J. digest. Dis.*, **10**, 247.
 — Almy, T. P., and Toscani, V. (1942). *Ann. intern. Med.*, **16**, 893.
 — and Weber, F. C. (1939). *Arch. intern. Med.*, **64**, 91.
 — (1940). *Ibid.*, **66**, 670.
 Wagener, H. P. (1945). *Coll. Pap. Mayo Clin.*, **37**, 414.
 Warren, S. (1930). Quoted by Ricketts (1947).
 White, P. (1946). *The Treatment of Diabetes Mellitus*, edited by E. P. Joslin and others, 1946, 8th ed., p. 741. London.
 — and Waskow, E. (1948). *Sth. med. J.*, **41**, 561.
 Young, F. G. (1937). *Lancet*, **2**, 372.
 — *et al.* (1949). *Nature, Lond.*, **164**, 209.

On May Day, 1950, the Archbishop of Canterbury inaugurated a festival at the Mayday Hospital, Croydon, and this year it was again celebrated. The patients were visited in the wards by civic, ecclesiastical, and other guests, and then a service was held in the hospital chapel. Afterwards there was a staff tea at which the hospital committee, doctors, nurses, and guests mingled in an informal manner. Many old members of the staff returned to the hospital to mark the occasion, and the day's events were rounded off by a social evening organized by the hospital sports clubs.